

YSTEM:OS - DIALOG OneSearch
File 155: MEDLINE(R) 1951-2005/Aug W1
(c) format only 2005 Dialog
File 55: Biosis Previews(R) 1993-2005/Aug W1
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File 34: SciSearch(R) Cited Ref Sci 1990-2005/Jul W5
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File 434: SciSearch(R) Cited Ref Sci 1974-1989/Dec
(c) 1998 Inst for Sci Info
File 340: CLAIMS(R)/US Patent 1950-05/Aug 09
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Set	Items	Description
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? s bh1 or bh2		
	412	BH1
	875	BH2
	S1	1036 BH1 OR BH2
? s bad		
	S2	37363 BAD
? s s1 and s2		
		1036 S1
		37363 S2
	S3	46 S1 AND S2
? s bcl?		
	S4	82651 BCL?
? s s3 and s4		
		46 S3
		82651 S4
	S5	46 S3 AND S4
? s heterodimer??		
	S6	44102 HETERODIMER??
? s s5 and s6		
		46 S5
		44102 S6
	S7	4 S5 AND S6
? rd		

>>> Duplicate detection is not supported for File 340.

>>> Records from unsupported files will be retained in the RD set.
... completed examining records
S8 3 RD (unique items)
? t s8/3,k,ab/1-3

8/3,K,AB/1 (Item 1 from file: 155)
DIALOG(R) File 155: MEDLINE(R)
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10840203 PMID: 7834748
Bad , a heterodimeric partner for Bcl -XL and Bcl -2, displaces Bax and promotes cell death.

Yang E; Zha J; Jockel J; Boise L H; Thompson C B; Korsmeyer S J
Howard Hughes Medical Institute, Department of Medicine, Washington University School of Medicine, St. Louis, Missouri 63110.

Cell (UNITED STATES) Jan 27 1995, 80 (2) p285-91, ISSN 0092-8674
Journal Code: 0413066

Contract/Grant No.: CA50239; CA; NCI

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

To extend the mammalian cell death pathway, we screened for further **Bcl**-2 interacting proteins. Both yeast two-hybrid screening and lambda expression cloning identified a novel interacting protein, **Bad**, whose homology to **Bcl**-2 is limited to the **BH1** and **BH2** domains. **Bad** selectively dimerized with **Bcl**-xL as well as **Bcl**-2, but not with Bax, **Bcl**-xs, Mcl-1, A1, or itself. **Bad** binds more strongly to **Bcl**-xL than **Bcl**-2 in mammalian cells, and it reversed the death repressor activity of **Bcl**-xL, but not that of **Bcl**-2. When **Bad** dimerized with **Bcl**-xL, Bax was displaced and apoptosis was restored. When approximately half of Bax was heterodimerized, death was inhibited. The susceptibility of a cell to a death signal is determined by these competing dimerizations in which levels of **Bad** influence the effectiveness of **Bcl**-2 versus **Bcl**-xL in repressing death.

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...Descriptors: physiology--PH; *Carrier Proteins--metabolism--ME; *Proto-Oncogene Proteins--metabolism--ME; *Proto-Oncogene Proteins c- **bcl**-2

Gene Symbol: **bad**

Chemical Name: Antibodies; **Bad** protein; Carrier Proteins; Macromolecular Substances; Proto-Oncogene Proteins; Proto-Oncogene Proteins c- **bcl**-2; Recombinant Proteins; **bcl**-x protein

8/3,K,AB/2 (Item 1 from file: 55)

DIALOG(R) File 55:Biosis Previews(R)

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0009667922 BIOSIS NO.: 199598135755

Bad, a Heterodimeric Partner for **Bcl**-X-L and **Bcl**-2, Displaces Bax and Promotes Cell Death

AUTHOR: Yang Elizabeth (Reprint); Zha Jiping; Jockel Jennifer; Boise Lawrence H; Thompson Craig B; Korsmeyer Stanley J

AUTHOR ADDRESS: Howard Hughes Med. Inst., Div. Mol. Oncol., Dep. Med., Washington University Sch. Med., St. Louis, MO 63110, USA**USA

JOURNAL: Cell 80 (2): p285-291 1995 1995

ISSN: 0092-8674

DOCUMENT TYPE: Article; Literature Review

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: To extend the mammalian cell death pathway, we screened for further **Bcl**-2 interacting proteins. Both yeast two-hybrid screening and lambda expression cloning identified a novel interacting protein, **Bad**,

whose homology to **Bcl** -2 is limited to the **BH1** and **BH2** domains. **Bad** selectively dimerized with **Bcl** -x-L as well as **Bcl** -2, but not with **Bax**, **Bcl** -x-s, **Mcl-1**, **A1**, or itself. **Bad** binds more strongly to **Bcl** -x-L than **Bcl** -2 in mammalian cells, and it reversed the death repressor activity of **Bcl** -x-L, but not that of **Bcl** -2. When **Bad** dimerized with **Bcl** -x-L, **Bax** was displaced and apoptosis was restored. When approximately half of **Bax** was heterodimerized, death was inhibited. The susceptibility of a cell to a death signal is determined by these competing dimerizations in which levels of **Bad** influence the effectiveness of **Bcl** -2 versus **Bcl** -x-L in repressing death.

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8/3,K,AB/3 (Item 1 from file: 34)
DIALOG(R) File 34:SciSearch(R) Cited Ref Sci
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06445160 Genuine Article#: YT745 Number of References: 31
Title: Dimerization properties of human BAD - Identification of a BH-3 domain and analysis of its binding to mutant BCL -2 and BCL -X-L proteins (ABSTRACT AVAILABLE)
Author(s): Ottolie S; Diaz JL; Horne W; Chang J; Wang Y; Wilson G; Chang S; Weeks S; Fritz LC; Oltersdorf T (REPRINT)
Corporate Source: IDUN PHARMACEUT INC,11085 N TORREY PINES RD/LA JOLLA//CA/92037 (REPRINT); IDUN PHARMACEUT INC,/LA JOLLA//CA/92037
Journal: JOURNAL OF BIOLOGICAL CHEMISTRY, 1997, V272, N49 (DEC 5), P 30866-30872
ISSN: 0021-9258 Publication date: 19971205
Publisher: AMER SOC BIOCHEMISTRY MOLECULAR BIOLOGY INC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814
Language: English Document Type: ARTICLE
Abstract: **Bad** , an inducer of programmed cell death, was recently isolated from a mouse cDNA library by its ability to bind to the anti-apoptotic protein **BCL** -2. Sequence analysis suggested that **Bad** was a member of the **BCL** -2 gene family that encodes both inducers and inhibitors of programmed cell death. To further analyze the role of **BAD** in the network of homo- and **heterodimers** formed by the **BCL** -2 family, we have cloned the human homologue of **BAD** and assessed its biological activity and its interactions with wild type and mutant **BCL** -2 family proteins. Our results indicate that the human **BAD** protein, like its mouse homologue, is able to induce apoptosis when transfected into

mammalian cells. Furthermore, in yeast two-hybrid assays as well as quantitative in vitro interaction assays, human **Bad** interacted with **BCL** -2 and **BCL** -X-L. Sequence alignments of human **BAD** revealed the presence of a BH-3 homology domain as seen in other **BCL** -2 family proteins. Peptides derived from this domain were able to completely inhibit the dimerization of **BAD** with **BCL** -X-L. Thus, as previously shown for **BAX**, **BAK**, **BCL** -2, and **BCL** -X-L, the BH3 domain of **BAD** is required for its dimerization with other **BCL** -2 family proteins. **BAD** was further analyzed for its ability to bind to various mutants of **BCL** -2 and **BCL** -X-L that have lost the ability to bind **BAX** and **BAK**, some of which retain biological activity and some of which do not. Surprisingly, all of the mutated **BCL** -2 and **BCL** -X-L proteins analyzed strongly interacted with human **BAD**. Our data thus indicate that mutations in **BCL** -2 and **BCL** -X-L can differentially affect the **heterodimeric** binding of different death-promoting proteins and have implications concerning the relationship between heterodimerization and biological activity.

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...Identifiers--PROGRAMMED CELL-DEATH; HOMOLOG BAK; APOPTOSIS; INHIBITION; **BCL** -X(L); GENE; DISTINCT; **BH1**

? log off

10aug05 09:17:30 User231882 Session D1461.2
\$1.00 0.295 DialUnits File155
\$0.21 1 Type(s) in Format 4 (UDF)
\$0.21 1 Types
\$1.21 Estimated cost File155
\$1.90 0.322 DialUnits File55
\$2.00 1 Type(s) in Format 4 (UDF)
\$2.00 1 Types

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$3.90 Estimated cost File55
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$2.08 Estimated cost File434
$3.05 0.175 DialUnits File340
$3.05 Estimated cost File340
OneSearch, 5 files, 1.195 DialUnits FileOS
$1.06 TELNET
$24.57 Estimated cost this search
$24.62 Estimated total session cost 1.406 DialUnits
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Logoff: level 05.06.01 D 09:17:30

You are now logged off